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Term:

(L7 or L6 or L5) and nitric oxide synthase

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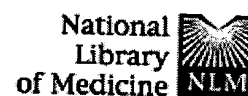
Cases

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result set

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<u>L9</u>	L8 AND angiogenesis	58	<u>L9</u>
<u>L8</u>	(L7 or L6 or L5) and nitric oxide synthase	80	<u>L8</u>
<u>L7</u>	taxol	2515	<u>L7</u>
<u>L6</u>	paclitaxel	1188	<u>L6</u>
<u>L5</u>	taxanes	812	<u>L5</u>
<u>L4</u>	L3 and nitric oxide	3	<u>L4</u>
<u>L3</u>	L2 and tubulin binding	72	<u>L3</u>
<u>L2</u>	taxol	2515	<u>L2</u>
<u>L1</u>	pacli\$5	1232	<u>L1</u>

END OF SEARCH HISTORY



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Search	Most Recent Queries	Time	Result
#44	Search #43 and Nos	13:20:22	<u>708</u>
#48	Related Articles for PubMed (Select 12643013)	13:18:18	<u>127</u>
#47	Search #43 AND angiogenesis	13:17:33	<u>8</u>
#46	Search #43 AND nitric oxide	13:17:15	<u>0</u>
#45	Search #43 AND Nos	13:17:01	<u>0</u>
#43	Search #42 AND combination therapy	13:16:19	<u>708</u>
#42	Search #25 AND clinical trials	13:15:41	<u>2879</u>
#35	Search #31	13:14:12	<u>127</u>
#34	Search #31 AND l-nma	13:09:17	<u>0</u>
#33	Search #31 AND neovasculature	13:08:16	<u>0</u>
#32	Search #31 and neovasculature	13:07:57	<u>127</u>
#31	Search #25 AND angiogenesis	13:06:23	<u>127</u>
#29	Related Articles for PubMed (Select 7537689)	13:03:34	<u>102</u>
#28	Search #25 AND nitroarginine	13:00:10	<u>1</u>
#26	Search taxol AND nitric oxide inhibitor	12:59:38	<u>8</u>
#25	Search taxol	12:55:53	<u>8517</u>
#22	Search taxol AND methylpyridine	09:17:19	<u>3</u>
#20	Search taxol AND nitroarginine	09:16:29	<u>1</u>
#19	Search taxol AND NO synthase inhibitor	09:15:26	<u>6</u>
#18	Search taxol	09:14:59	<u>8517</u>
#16	Related Articles for PubMed (Select 10197639)	09:13:35	<u>182</u>
#15	Search #14 AND tubulin	09:13:18	<u>3</u>
#14	Search nitric oxide synthase inhibitor AND cancer treatment	09:12:39	<u>130</u>
#13	Search nitric oxide synthase inhibitor	09:12:25	<u>8467</u>
#3	Search combretastatins	09:10:15	<u>23</u>
#8	Search #3 AND NO	09:07:09	<u>0</u>
#7	Search #3 AND nitric oxide	09:07:03	<u>0</u>

#6 Search #3 AND nitric oxide synthase	09:06:53	<u>0</u>
#5 Search #3AND nitric oxide synthase	09:06:44	<u>23</u>
#2 Search combretstatins	08:56:25	<u>0</u>
#1 Search combrestatins	08:56:05	<u>0</u>

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Colchicine Induces Apoptosis in Cerebellar Granule Cells

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Available online 26 April 2002.

Abstract

Exposure to 1 μ M colchicine, a microtubule disrupting agent, triggered apoptosis in rat cerebellar granule cells (CGC). Apoptotic nuclei began to appear after 12 h followed by oligonucleosomal DNA laddering, whereas inhibition of the mitochondrial 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazoliumbromide metabolism became significant between 18 and 24 h, when most cells already had apoptotic nuclei. These events were preceded by loss of tau protein and fragmentation of α and β tubulins. Colchicine treatment also caused alterations in Ca^{2+} responses to chemical depolarization and a moderate, but progressive, increase in the resting intracellular Ca^{2+} concentration. Nearly all neurons expressed c-Fos after the treatment with colchicine. However, while in part of the cell population c-Fos levels subsequently declined, in the neurons undergoing apoptosis the protein was still expressed, but had an abnormal intracellular localization. An increased expression of the constitutive nitric oxide synthase (NOS-I) was also detected at 12 h and was followed by increased nitrite production. Treatment with 100 nM taxol to stabilize the microtubuli prevented DNA laddering and apoptotic body formation induced by colchicine. In contrast, pretreatment with the *N*-methyl-D-aspartate receptor-antagonist, MK-801, or L-type Ca^{2+} channel blockers did not prevent colchicine-induced CGC

apoptosis. Inhibitors of NOS were also ineffective in preventing apoptotic body formation and DNA laddering, whereas they delayed the secondary cell lysis. These results support the idea that colchicine-induced cytoskeletal alterations directly initiate the genetic and structural modifications that result in CGC apoptosis.

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